Persistence of Recruitable Coronary Collaterals in the Absence of Coronary Vasospasm in a Patient with Variant Angina

Kazuhito Yamashita, Masaaki Takeuchi, Yasuhide Nakashima

Second Department of Internal Medicine, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishiku, Kitakyushu, Fukuoka 807, Japan

Abstract

Recruitable coronary collaterals may appear when spasm suddenly occludes the coronary artery. We report a patient with variant angina who had visible collateral vessels on a control coronary angiogram, despite the presence of normally appearing coronary arteries. These collaterals disappeared after intracoronary administration of nitroglycerin. These findings suggest that recruitable collateral vessels can remain patent long after spontaneous attacks of angina have resolved, and become visible when there is a pressure difference between two small coronary arteries.

Key words: Recruitable collateral vessels—Vasospastic angina—Coronary angiography

Collateral coronary vessels may develop when the artery becomes severely narrowed or occluded. In patients with vasospastic angina but without critical stenosis of a coronary artery, coronary collaterals can be visualized only when the parent vessel is subtotally or totally occluded [1]. We report a patient with vasospastic angina in whom collateral vessels were seen on the control coronary angiogram. Those vessels disappeared after the intracoronary administration of nitroglycerin. To our knowledge, there is no report of a patient who had visible collateral vessels on a control coronary angiogram, despite the presence of normal coronary arteries.

Case Report

A 61-year-old Japanese man with no history of angina showed an elevation of the ST segment on the electrocardiogram (ECG) taken immediately after completion of a prostatectomy. This electrocardiographic change disappeared after the administration of nitroglycerin. The diagnosis was variant angina and the patient was given antianginal medications but continued to have chest pain at rest two or three times a week. He was then transferred to our department for treatment. The ECG recorded at rest showed a normal sinus rhythm with normal axis deviation. There were no abnormal Q waves or ST-segment changes. Treadmill exercise ECG showed no significant ST-segment changes but the patient complained of chest oppression. Results of exercise thallium-201 single-photon emission computed tomography (T1-201 SPECT) were normal and the 123Iodine metaiodobenzylguanidine single-photon emission computed tomography (123I-MIBG SPECT) demonstrated no defect or reduction in uptake of radioactive iodine in the left ventricular wall. Twelve-lead ECGs, recorded continuously while the patient was experiencing chest pain, were normal.

Based on these findings, we discontinued the antianginal regimen and after 1 week we performed cardiac catheterization to reveal the details of coronary anatomy, including anatomic or functional pathology. The patient had no episodes of chest pain the day of cardiac catheterization, and ECG recordings performed in the catheterization laboratory were normal. The left coronary angiogram revealed a mild increase in the tonus of the epicardial coronary arteries, but no significant stenosis (Fig. 1A, B). A right coronary angiogram also showed no significant stenosis but we observed collateral vessels from the right coronary artery to the first diagonal branch (Dx) of the left anterior descending artery (LAD) (Fig. 1C, D). Before the injection the catheter tip pressure was not reduced, and there was reflux of contrast medium into the aorta before the injection was terminated. At that time the patient was free of chest pain and exhibited no ST-T changes on 12-lead ECG. After intracoronary administration of acetylcholine 50 ~g into the left coronary artery the patient complained of chest pain. The ST segment was elevated in leads I, V4, and V6, and depressed in leads II, III, and V2. The distal LAD and Dx and the proximal left circumflex artery were completely occluded. Chest pain disappeared after intracoronary administration of nitroglycerin and the ECG returned to baseline. Coronary angiography revealed no stenosis in the left coronary artery and the collateral vessels from the right coronary artery to the first diagonal branch were no longer visible (Fig. 2). A left ventriculogram showed hypokinesis of the anterolateral, apical, and septal walls.

Discussion

Under physiological conditions, coronary collateral vessels are usually present between intracoronary arteries and between the different coronary arteries. However, such vessels are generally less than 40 ~m in diameter and are not visible on angiograms. The development of collateral vessels is reportedly enhanced by exercise, severe anemia, and gradual coronary occlusion [2]. Collateral vessels are often found in the presence of more than 90% stenosis of the epicardial coronary artery. The development of collaterals depends upon the course of coronary stenosis. Several studies report that the presence of angiographically visible coronary collaterals results in a greater preservation of left ventricular function.
with a reduction in infarct size [3]. Patients with coronary collaterals show a lower incidence of late aneurysm formation after a myocardial infarction [4].

Coronary collateral circulation may develop after repetitive coronary spasms even in the absence of significant organic stenosis. Matsuda et al. [5] reported that the coronary artery, distal to the total occlusion induced by coronary spasm, received collaterals from the nonspastic artery in 7 of 11 patients who had variant angina with no more than 70% stenosis in any coronary artery. In experimental studies, repeated brief (2 min) episodes of ischemia every 30 min increased the development of the collateral vessels in conscious dogs without significant coronary stenosis [6]. To our knowledge there are no previous reports of recruitable collateral vessels that were visible on a control angiogram in the absence of vasospasm or significant stenosis of the epicardial coronary arteries.

Takano et al. [7] have demonstrated that a regional uptake mismatch between T1-201 SPECT and $^{123}$I-MIBG SPECT can be observed in patients with coronary vasospasm, indicating a regional myocardial sympathetic dysinnervation which is specifically located in the distribution of vasospasm-induced vessels. Although we did not observe regional myocardial sympathetic dysinnervation in our patient, the reduced wall motion in the territories of the LAD suggested that the repeated episodes of coronary spasm had led to myocardial stunning.

A proposed explanation for the findings reported here is that repeated coronary vasospasm, that had not been clinically de-